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AIR POLLUTION ASPECTS OF BERYLLIUM AND ITS COMPOUNDS

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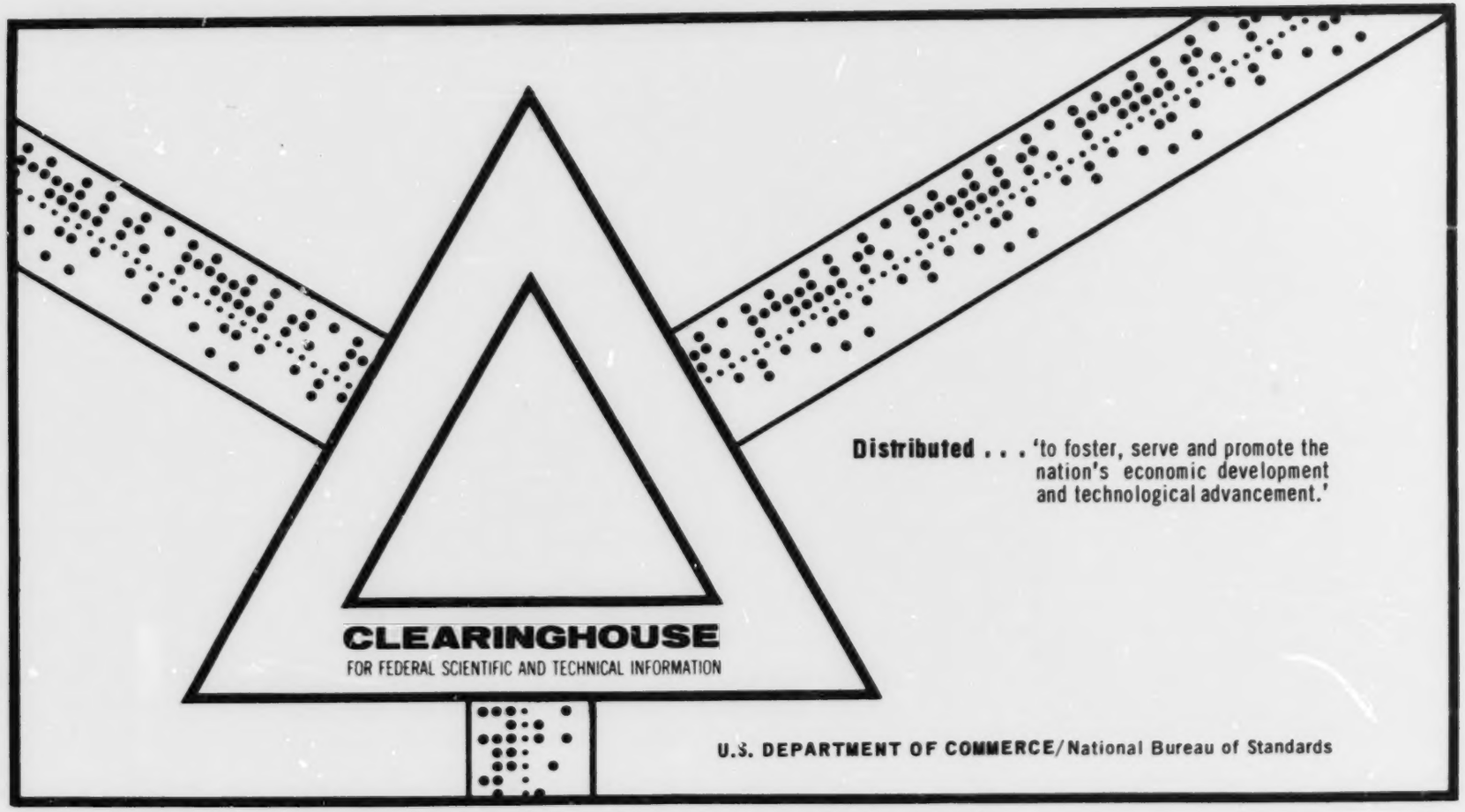
Litton Systems, Incorporared
Bethesda, Maryland

September 1969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AIR POLLUTION ASPECTS

OF

BERYLLIUM AND ITS COMPOUNDS

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Service
Department of Health, Education, and Welfare
(Contract No. PH-22-68-25)

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September 1969

FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds (includes chromic acid)
Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

Inhalation of beryllium (Be) or its compounds by humans or animals can cause a body-wide systemic disease, with pulmonary damage being of major concern. The acute form occurs as a chemical pneumonitis, with inflammation of the mucosa and submucosal tissues of the respiratory tract. Chronic beryllium disease, which differs clinically from the acute case, has also caused severe respiratory damage. As of 1965, 735 cases of beryllium disease had been recorded in the Beryllium Case Registry, with a fatality rate of approximately 26 percent. Bone and lung cancers have been produced experimentally in animals, and 20 malignant tumors have been recorded among the 735 cases of beryllium disease; however, the available evidence is not considered sufficient to positively incriminate beryllium as a carcinogen in humans.

There is some evidence that beryllium in soils is toxic to plant life; no evidence has been found on the effects of atmospheric beryllium on plants or materials.

The major sources of beryllium in the atmosphere are the industrial processing plants engaged in the extraction, refining, machining, and alloying of the metal. The combustion of coals containing small quantities of beryllium, and the proposed use of beryllium as an additive in rocket fuels, are also possible sources.

Conventional air-cleaning procedures have been generally adopted by the beryllium industry, producing marked reductions

in the amount of beryllium emitted to the atmosphere. Data on the costs of abatement are limited; one study indicated that costs for control amount to approximately 20 percent of the cost of normal operation. No data were found on the costs of damage produced by beryllium air pollution; however, legal actions are currently in progress in Pennsylvania courts which may provide data on values of health impairment resulting from beryllium exposure.

Methods for analysis of beryllium in the atmosphere are available; however, they do not discriminate between the various compounds of beryllium.

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1. INTRODUCTION

Beryllium and its compounds, when present in the environmental air, are of concern because of their effect on the health of humans and animals, since beryllium is among the most toxic and hazardous of the nonradioactive substances being used in industry. Beryllium is commonly found as an atmospheric pollutant within the confines and in the proximity of industrial plants producing or using beryllium substances.

Almost all the presently known beryllium compounds are acknowledged to be toxic in both the soluble and insoluble forms, depending on the amount of material inhaled and the length of exposure. Soluble beryllium compounds, such as beryllium sulfate and beryllium chloride, commonly produce acute pneumonitis; insoluble compounds, such as metallic beryllium and beryllium oxide, can produce chronic pulmonary disease (berylliosis).[2] However, it should be noted that the toxic beryllium effect is not limited to berylliosis but instead is a body-wide systemic disease.

Expanded industrial use of beryllium in the 1930's, particularly the large-scale production of fluorescent lamps using phosphors of beryllium oxide, produced a number of cases of pulmonary diseases which were initially attributed to causes other than exposure to beryllium. Recognition of the toxic effects of beryllium, however, beginning in 1943, resulted in establishment of the first Community Air Limit

for metal substances, as well as in health safety procedures which were exceptionally effective in controlling further occurrences of the disease. However, increased use of beryllium in the metallurgical industry, along with its proposed use as a high-energy fuel for rocket motors, suggests that study should be made of the air pollution aspects of this highly toxic material.

2. EFFECTS

Claims and counter-claims concerning the toxicity of beryllium and its compounds have been made as early as the latter part of the 19th century; however, serious study of this problem did not commence until beryllium poisoning was reported in humans by Weber and Englehardt in 1933.[61] Health problems arising among workers engaged in the production and manufacturing of beryllium and its products during the late 1930's and 1940's led to case studies and research which definitely established the toxic nature of beryllium in most of its physical and chemical forms. The properties, toxicity, and uses of beryllium and certain of its compounds are listed in Table 12 of the Appendix.

2.1 Effects on Humans

The major hazard from beryllium arises from the inhalation of beryllium or its compounds.[52] Damage to the skin and mucous membranes can occur from handling of the soluble salts of beryllium. These manifestations can be cured completely, but may last for considerable periods of time. The effects of beryllium intoxication can be mild, moderate, or severe, and can even prove fatal when severe damage to the pulmonary system occurs.

2.1.1 Acute Beryllium Disease

2.1.1.1 Respiratory Effects

Acute chemical pneumonitis has been produced by inhalation of virtually all beryllium compounds. These include

beryllium metal, beryllium oxide, beryllium sulfate, beryllium fluoride, the hydroxide, and the chloride. Responses to beryllium exposure range from transient rhinitis, pharyngitis, or tracheobronchitis to severe pulmonary reaction. The degree of response seems to be dependent upon the degree, duration, and type of exposure.[21] Rapidly fatal cases have resulted from exposure to large concentrations of soluble salts in beryllium-processing industrial plants. However, most commonly the illness is insidious in nature, developing as a dry cough and progressing to substernal discomfort and pain, general weakness and fatigue, and loss of weight.[11,21,32,62] All regions of the upper respiratory tract may be affected, with acute inflammation of the mucosa and submucosal tissues.[14] Lung changes may develop in 1 to 3 weeks, and are characterized by chest X-ray haziness, progressing to appearance of discrete or conglomerate nodules. Cases of acute pneumonitis are usually hospitalized for observation and treatment, to include bed rest, use of oxygen bronchodilators, antihistamines, antibiotics, and cortisone.[32] Other cases of a less severe nature, not requiring hospitalization, may not receive medical attention, nor will they be included in medical statistics.

According to Eisenbud, as reprinted in Reference 46 (1958), the disease runs its course in a matter of weeks, and unless death occurs, complete recovery results. This statement as to the course of the disease has been disputed by

Hardy,[25] who has stated that no medical follow-up was made of workers discharged for having "any symptoms whatsoever of toxic beryllium effect." She also notes the long residence of beryllium in the body and the possibility of chronic disease occurring at a much later period of time following exposure.[25] Cholak, et al.[11] state that while some patients with acute beryllium pneumonitis have also acquired the chronic form, a sequential relationship between the two forms of the disease has not been established. This view is reinforced by the Committee on Toxicology[2] of the National Academy of Sciences—National Research Council, which stated in 1966 that "As far as can be determined, there are no cases of uninterrupted progression from acute chemical pneumonitis to chronic beryllium disease from a single exposure." De Nardi et al.[14] reported in 1953 that in a large series of cases, 47 survivors of the acute disease were observed for as long as 12 years, with no occurrence of the chronic disease in any person. On the other hand, it has been stated that when these survivors were studied further, a number of them were found to have chronic disease.[25] Hardy[23] also reported in 1965 that on the basis of data from the Beryllium Case Registry of June 1964, 46 cases out of 725 (6.3 percent) progressed from acute to chronic and the mortality was high.

The toxicity of beryllium and its compounds is not clearly defined;[27,29,46] measurements of concentrations

and particle size are not always available or adequate for definite medical conclusions. For instance, concentrations of beryllium oxide of 30,000 $\mu g/m^3$ produced no acute cases in exposed employees of one plant, whereas in a second plant, exposure to 4,000 $\mu g/m^3$ produced a high incidence of acute disease, associated with a high incidence of fatalities.[46] In this regard, laboratory studies have revealed that the toxicity of beryllium oxides produced at 1,600°C is considerably less than that produced at relatively low temperatures (500°C).[46] These findings were confirmed by laboratory tests showing that animals were able to tolerate high exposures to oxides formed at high temperatures but developed acute poisoning when exposed to similar concentrations of low-fired oxides.[52] Studies (1951) of the occurrence of the acute disease in beryllium plants revealed that all cases had occurred after exposure to concentrations in excess of 100 $\mu g/m^3$, and when concentrations of the soluble beryllium compounds exceeded 1,000 $\mu g/m^3$, acute disease occurred in almost all personnel.[19] Unfortunately, the time period for exposure to these concentrations was not known or specified.

The Panel on Toxicity of Beryllium of the Materials Advisory Board, National Academy of Sciences—National Research Council reported[46] (1958) that no acute illnesses had occurred when peak concentrations did not exceed 25 $\mu g/m^3$. It was further reported that inhalation of 40 $\mu g/m^3$ of beryllium

sulfate by experimental animals produced typical lung damage in some—but not all—animals; no damage was noted from exposure to concentrations of 4 $\mu g/m^3$.

The extent of involvement of the respiratory tract, the degree of severity, and the causative factors of acute disease have been tabulated by the Brush Beryllium Company,[63] based on observations on selected employees of this company. These data are presented in Table 1.

TABLE 1

ACUTE RESPIRATORY PROBLEMS RESULTING FROM BERYLLIUM EXPOSURE[63]

Type	Degree of Severity	Causation	Comments
1. Nose and throat (naso-pharyn-gitis)	Minor	Fumes, mists, and/or dusts of soluble salts of Be. Usually result of minor-degree exposure during massive incident	BeF_2 most reactive of all beryllium compounds
2. Bronchial tubes (tracheo-bronchitis)	Inter-mediate	Usually the result of relatively high level of exposure over prolonged period of time (2 weeks +)	More commonly experienced with $BeSO_4$ than with other compounds
3. Lungs (acute beryllium chemical pneumo-nopathy)	Major	Result of very high level of exposure in relatively short time	BeF_2 more reactive as to severity and morbidity than other compounds

2.1.1.2 Other Physiological Effects

Acute beryllium poisoning can produce skin, eye, and other limited physiological reactions. Skin problems are usually associated with contact exposure or implant of the soluble salts of beryllium. Table 2 from data of the Brush Beryllium Company [63] lists the principal types of skin problems, their causes, relative severity, and frequency (on a scale of 0 to 4, with 4 representing the maximum intensity). Lesions of the skin are similar to those produced by other primary irritants; dermatitis occurs most frequently in the exposed portions of the body, usually with symptoms of pruritus and sensations of burning. The affected areas usually improve after termination of exposure. Healing is assisted by application of aluminum acetate soaks.

Mucous membranes of the conjunctiva, nose, nasopharynx, trachea, and bronchi may be affected by the soluble salts of beryllium. Acute responses may occur as hyperemia, edema, ulceration, hemorrhage, and such symptoms as itching, burning, and rhinorrhea.[11] Tracheobronchitis may appear rapidly or insidiously, with throat irritation, cough, tightness of chest, and other symptoms difficult to distinguish from those of acute beryllium pneumonitis.[11]

Hazard,[26] as discussed by Vorwald,[60] reported mild thickening of the right ventricular wall of the heart in two cases of acute beryllium disease. Moderate inflammation of the

TABLE 2

ACUTE SKIN PROBLEMS RESULTING FROM BERYLLIUM EXPOSURE[63]

Type	Cause	Causal Frequency and Severity			Comments
1. Contact dermatitis (primary irritant)	Undue contact with soluble salts of Be	BeF_4 $BeSO_4$ $BeCl_2$ $Be(NO_3)_2$ $Be(OH)_2$	4+ 2+ ? ? 0	4+ 2+ 2+ ? 0	Gross aspect varies with the salt involved
2. "Allergic" dermatosis (angioneurotic edema with superimposed primary irritant)	Fumes, mists and/or dusts of BeF_2 Equivocal with $BeSO_4$	BeF_2 $BeSO_4$ $Be(OH)_2$	1% ?		
3. Chemical ulcer	Contamination of neglected minor cut with soluble salts of Be	BeF_2 $BeSO_4$ $Be(OH)_2$	4+ 2+ 0	4+ 2+ 0	Most frequently over bony structure, e.g., knuckle
4. Ulcerating granuloma	Implant of soluble salt of Be	$BeSO_4$ BeF_2 $Be(OH)_2$	4+ 1+ 0	2+ 4+ 0	Existence of granuloma not verified with BeF_2

liver was also noted as well as some evidence of severe central necrosis, focal hemorrhage of the spleen, and mild edema of the brain.

2.1.2 Chronic Beryllium Disease

Histopathologic lesions caused by chronic beryllium disease occur in the skin, subcutaneous tissues, and the lungs. Lesions in the skin and subcutaneous tissues can be cured by minor surgery. Granulomatous reactions in the lung can produce severe and permanent respiratory damage or death.[11] It is believed that most all of the systems of the body can be affected; there is evidence that beryllium affects enzyme systems, and is not limited to local effects in the lungs. Johnson[30] has pointed out that the granulomatous nodules are a prominent histological feature but do not account for the respiratory embarrassment responsible for much of the disability associated with this disease. Instead, there is a biochemical lesion—a defect in the mechanism for transport of oxygen.

2.1.2.1 Respiratory Effects

Although berylliosis and beryllium granulomatosis have been widely used designations for chronic beryllium disease, these are misnomers, according to Johnson,[30] for what is basically a systemic disease, with recognizable beryllium injury appearing in many tissues and organs. Every organic system would appear to be involved in response to beryllium inhalation, except organs in the pelvic area. Since beryllium has a long residence time in the body, it may have a potential for cancer induction, as has been suggested by animal experiments.

Attempts to correlate the chronic effects with exposure

have been unsuccessful.[21] The delay in onset of manifestations can vary from months to as long as 23 years.[2,32] The illness is of long duration, with a high mortality rate. Cases have occurred in persons subject to exposure to beryllium in many forms in a wide variety of occupations, and in persons not connected with the processing of beryllium but residing in the neighborhood of beryllium plants. The evidence also indicates that members of households of workmen employed in beryllium plants have contracted this disease from exposure to dust carried home on clothing.[12]

According to the National Academy of Sciences—National Research Council,[2] "The pathogenesis of chronic beryllium disease is still unknown. The fact that only a small percentage of the persons exposed develop the disease lends support to the hypothesis advanced by several investigators that there is an immunological abnormality associated with the disease, or that the susceptibility is in some way related to an inborn error of metabolism." However, a recent study (1968) would appear not to support the immunologic view contained in this report. In this study by the Aerospace Medical Research Laboratories, two beagle dogs were exposed for 20 minutes by respiratory route to rocket exhaust products containing a mixture of beryllium oxide, beryllium fluoride, and beryllium chloride at an average beryllium concentration of 115,000 $\mu g/m^3$. Robinson *et al.*[47] concluded, "It appears that the

lesions in these dog lungs are more typically the classical reactions to a foreign body than immunologic in character."

The Council further states, "Among the factors associated with the chronic disease that have complicated the interpretation of the epidemiological studies are the following: the extremely small amounts of beryllium-containing materials alleged to have produced the disease; the well-documented fact that many persons have received severe exposures to beryllium-containing compounds without developing the disease; the latent period between exposure and symptoms; the occurrence of illness in only a small percentage of the exposed population; the similarity between chronic beryllium disease and other chronic diffuse pulmonary diseases; the uncertainty as to the identity of the physical-chemical properties of the beryllium compounds involved; and the lack of any quantitative data on the magnitude of the exposure and particle size."

This source also considers that the chronic form of the disease is caused by exposure to the insoluble compounds of beryllium, and that the few chronic cases alleged to have resulted from exposure to soluble compounds could actually have been due to either unknown exposure to insoluble compounds or to hydrolysis in the tissues of beryllium hydroxide or oxides.

As of June 1966, 498 cases of chronic beryllium disease had been recorded in the Beryllium Registry. An analysis of 460 of these cases[2] noted that over 400 were industrial cases

while 60 were nonindustrial. Hall et al.,[20] in an analysis in 1959 of 382 cases from the Beryllium Case Registry, reported the symptoms of chronic disease as dyspnea (69 percent), loss of weight (61 percent), cough (78 percent), increased fatigue (34 percent), pains in the chest (31 percent), loss of appetite (26 percent), and general weakness (17 percent).

Little quantitative data are found in the literature concerning atmospheric concentrations resulting in chronic beryllium poisoning. Qualitatively it is reported that chronic cases have occurred as a result of single massive doses of beryllium-contaminated air[9] as well as from prolonged exposure to concentrations of only a few micrograms per cubic meter.[52] Sterner and Eisenbud[52] have reported neighborhood cases in which chronic disease occurred in approximately 1 percent of the population exposed to concentrations of 1 $\mu g/m^3$ within one-fourth mile of a beryllium plant. However, among workers within this same plant exposed to concentrations as much as 1,000 times greater, only 0.5 percent contracted the disease. Failure to recognize the symptoms of beryllium poisoning may have contributed to an underreporting of this disease.

2.1.2.2 Other Physiological Effects

Chronic disease of the skin as a result of beryllium exposure is relatively rare, but highly publicized as a result of fluorescent tube breakage and in-plant accidents. It occurs infrequently since the discontinuance, in 1949, of the use of

beryllium phosphors in fluorescent tube manufacturing.[63] However, Breslin[9] believes contact dermatitis to be a significant disease problem still in the beryllium extraction industry, where massive quantities of sulfate and fluoride compounds are processed.

Soluble salts of beryllium cause contact dermatitis as well as conjunctivitis. The dermatitis can progress to ulceration. Conjunctivitis, resulting from accidental splashes of beryllium salts, is similar to inflammations caused by other irritants.

2.1.2.3 Community Episodes

According to Lieben,[36] "Beryllium disease is to date (1961) the only generally accepted chronic disease entirely due to air contamination of the community atmosphere." (However, there are reported cases of manganese pneumonia in Scandinavia and other neighborhood cases of asbestos malignancies.[25]) Eisenbud et al.,[16] Lieben,[34] and Lieben et al.[56] reported on the following cases of community contamination:

(1) Northern Ohio, 1948-1949.[16,36] A total of 11 chronic cases of beryllium disease occurred in the neighborhood of a beryllium extraction plant in northern Ohio. The persons involved resided in the area of the plant, but none were employed in or near the plant. Five of these persons lived within one-fourth of a mile from the plant, three within half a mile, two within three-fourths of a mile, and one within

2 miles. Since 500 persons lived within a quarter-mile of the plant, the prevalence of disease within that distance approximated 1 percent as of 1961. Air sampling in the vicinity of the plant showed concentrations ranging from 0.000 to 2,100 $\mu g/m^3$ at distances of 750 feet from the plant; average concentrations at the three-fourth mile radius ranged from 20 to 40 $\mu g/m^3$.

(2) Pennsylvania, 1947-1959.[36,56] Twenty-six cases of chronic poisoning occurred in the vicinity of a beryllium refinery and alloy fabricating plant in Pennsylvania, with onset of symptoms occurring over a period of 12 years. This particular plant, covering 15 acres, is located in a shallow valley through which prevailing winds are channelled—from the northeast 9 months of the year, and from the southeast the remaining 3 months. Most of the cases occurred in the downwind sectors from the plant: one within 0.7 miles, eight within 2 miles, and the remainder within 3 to 6 miles. Extensive sampling of the atmospheric concentrations, commenced in 1958, revealed a mean concentration of 0.015 $\mu g/m^3$ in the area of the plant. Ten percent of the 500 samples showed concentrations in excess of 0.035 $\mu g/m^3$. Also noteworthy was the aftermath of a 2-week shutdown of the plant, which resulted in a reduced mean concentration level of 0.0015 $\mu g/m^3$. Sussman,[56] in his comments about this reduced level, mentioned the possibility that beryllium deposited on the ground and other surfaces in

the area might become airborne. Background levels of beryllium, measured by Sussman elsewhere in the State, averaged 0.0002 $\mu g/m^3$.

Other neighborhood cases of less dramatic impact have been reported earlier by Gelman[18] and Hardy.[22]

2.1.3 Carcinogenicity

The Committee on Toxicology of the National Academy of Sciences—National Research Council[2] reports (1966): "While certain beryllium salts and oxides have been productive of osteogenic sarcomas in rabbits following intravenous administration and primary lung tumors in rats and monkeys following inhalation, there is no evidence that community or industrial exposure to beryllium compounds is associated with an increase in the incidence of cancer in humans."

According to Hardy,[23] there were (in 1966) 20 malignant tumors recorded among the 735 Registry cases; five of these are lung cancer, and one a bone tumor. Without knowledge of population at risk or more complete data, it is impossible to incriminate beryllium as a carcinogen in human beings. However, beryllium has a long residence time in the body and therefore may have a cancer-producing effect.

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

No information has been found in the literature on the effects of beryllium or its compounds on domestic or commercial

animals. It is possible that accidental poisoning of animals has occurred simultaneously with the instances of "neighborhood cases" of beryllium poisoning in humans. However, the prominence accorded to human cases, and the volume of data attending these cases, undoubtedly so overshadowed the animal cases that they received no mention in the literature.

The Community Air Quality Guide for Beryllium, established by the American Industrial Hygiene Association,[13] states that "no potential harmful effects are reported for animal exposures at outdoor air concentrations."

2.2.2 Experimental Animals

Recognition of the toxic effects of beryllium and the increasing incidence of beryllium disease in and around industrial plants resulted in a vast program of experimental research to identify and quantify the toxic characteristics of beryllium and its various compounds. A large amount of research employing laboratory animals has been recorded in the literature; an excellent summarization of this work is contained in the chapter entitled "Experimental Beryllium Toxicology,"[60] written by Vorwald, Reeves, and Urban for the text Beryllium—Its Industrial Hygiene Aspects, edited by Stokinger. Much of the following discussion is condensed therefrom, unless otherwise noted.

2.2.2.1 Acute Beryllium Disease

In order to understand the nature of beryllium pneumonitis in humans, many experiments were conducted with reference

to the effect of beryllium poisoning on the respiratory system. Marradi-Fabroni[37] observed acute and subacute bronchiolitis and pneumonitis in guinea pigs exposed to atmospheres of beryllium carbonate. From these observations he concluded that beryllium was the cause of the disease he labelled "berylliosis."

In 1947, Sprague et al.[51] reported on experiments with 56 animals—2 dogs, 3 rabbits, 10 rats, 14 guinea pigs, 20 mice, and 7 hamsters—exposed to beryllium sulfate dusts and beryllium metal fumes. In atmospheric concentrations of 800,000 $\mu g/m^3$, mice died within 11 days, while dogs, rabbits, and guinea pigs survived; however, acute damage was observed in the lungs, liver, and kidneys. High concentrations, in the order of 24,000,000 $\mu g\text{-}min/m^3$,* produced pulmonary hemorrhage and death, but at low concentrations (4,000,000 $\mu g\text{-}min/m^3$) mice survived exposures for 60-day periods and developed tolerance to 10 times the otherwise lethal dose.

Stokinger et al.[54,55] reported on observations of approximately 500 animals exposed to concentrations of 100,000, 50,000, 10,000, and 1,000 $\mu g/m^3$ of beryllium sulfate mist. The general results of these experiments are tabulated in Table 3.

In experiments designed to determine whether inhaled insoluble compounds would produce acute pneumonitis, Hall et al.[19] in 1950 exposed six different species of animals to

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*$\mu g\text{-}min/m^3$ = microgram-minutes per cubic meter.

TABLE 3

RESULTS OF EXPOSURES OF BERYLLIUM SULFATE HEXAHYDRATE[54,55]

Concentration ($\mu g/m^3$)	Total Exposure (hours)	Species of Animal	Effects
100,000	66	Laboratory rodents plus monkeys, goats, dogs, cats, and chickens	Lethal to majority of animals
50,000	234	"	" " "
10,000	426	"	LD_{50} for rats; typical pneumonitis in most species
1,000	426	"	Detectable injury in some species

aerosols of different grades of beryllium oxide dust at mean concentrations of 85,000 $\mu g/m^3$ over periods of 6 hours daily, 5 days per week, for 10 to 17 days. All oxides produced acute pneumonitis. In one experiment, four female dogs were exposed to 10,000 $\mu g/m^3$ of low-fired oxide for 40 days; this concentration also resulted in acute disease.

Vorwald et al.[60] concluded in their review that certain forms of beryllium, even in extremely small amounts, are capable of producing an acute and often lethal inflammatory reaction in the lung.

Altman et al.[5] have compiled data on the physiological changes and mortality occurring in mammals and birds from the inhalation of beryllium fluoride and beryllium sulfate. These data are presented in Table 13, Appendix.

2.2.2.2 Chronic Beryllium Disease

Efforts to produce symptoms of chronic beryllium poisoning in experimental animals were largely unsuccessful until experimental procedures were followed which closely resembled exposure conditions under which this disease was contracted by humans. This required an extensive experimental period involving low concentrations of the pollutant, principally the insoluble oxide. Prine et al.[44] reported on experiments inducing chronic beryllium disease in dogs, using beryllium oxide and metallic beryllium, and noted the interesting fact that two of the six animals exposed for 30 to 36 months developed the characteristic pulmonary granulomas.

In a similarly lengthy experimental period, Vorwald[59] exposed a group of rhesus monkeys to an atmospheric concentration of 35 $\mu g/m^3$ of beryllium sulfate aerosol. The experiments were continued for many months, with only occasional periods of respite from the beryllium atmosphere as necessary to insure the continued survival of the animals. Laboratory examinations subsequent to the death of the animals revealed that the beryllium compounds had produced symptoms of widespread chronic beryllium pneumonitis with associated granulomas. Also, the animals had developed primary bronchopulmonary cancer. In a comment on this experiment, Vorwald et al.[60] state, "With respect to the inflammatory pulmonary changes in both the rat and the monkey, the response is judged to be the respective

animal's counterpart of the chronic beryllium pneumonitis as seen in some human subjects after their exposure to toxic beryllium compounds."

2.2.2.3 Carcinogenicity

As mentioned briefly in the preceding section, pulmonary cancer was developed in monkeys by Vorwald et al.[60] Case histories include mention of cancerous growths created by intrabroncheal injection of beryllium oxide and by inhalation of beryllium sulfate aerosol. Exposures to average concentrations of 35 $\mu g/m^3$ intermittently over the course of more than 5 years resulted in death in two of the 10 monkeys. These experiments were still in progress as of 1966.

In these experiments, conducted by the same authors subsequent to their previous work with rats, proliferations of pulmonary epithelial cells were induced by either the beryllium oxide or the sulfate. Primary pulmonary cancer appeared as early as 8 months after intratracheal injection and 9 months after inhalation. The incidence of cancer was nearly 100 percent in a group of 150 rats exposed daily for 13 months to beryllium sulfate aerosol in concentrations of 21 to 42 $\mu g/m^3$. Even at the very low concentration of 2.8 $\mu g/m^3$, 13 of 21 rats developed pulmonary cancer.[45]

2.3 Effects on Plants

There is some indication that beryllium is toxic to plant life. Romney, Childress, and Alexander[48] investigated

the effects of beryllium on bush beans, concluding that it was toxic in levels in excess of 1 ppm in soil solution. This conclusion was based on tests of bush-bean growth in nutrient solutions containing 0.0, 0.5, 1.0, 2.0, 3.0, and 5.0 ppm of beryllium. Reduced growth rates were noted at the increased beryllium concentrations.

Hopkins[43] also noted the toxic nature of beryllium sulfate on the growth of seedling roots.

According to the Community Air Quality Guide for Beryllium,[13] no harmful effects to vegetation at outdoor air concentrations have been reported.

2.4 Effects on Materials

No harmful effects to materials were reported in the literature resulting from beryllium or its compounds in the atmosphere.

According to the Community Air Quality Guide for Beryllium, no reports of permanent materials damage or soiling effects due to beryllium air pollution at outdoor air concentrations are known.

2.5 Environmental Air Standards

Environmental Standards for atmospheric concentrations of beryllium were first proposed in 1948 by a Beryllium Advisory Committee of the U.S. Atomic Energy Commission. In 1949, the AEC adopted these recommended standards, which are summarized below:[11]

(1) The in-plant concentration of beryllium should not exceed 2 $\mu g/m^3$ as an average concentration throughout an 8-hour day.

(2) An absolute concentration limit of 25 $\mu g/m^3$ should not be exceeded for any period of time, however short.

(3) Community air (outside of plants handling beryllium compounds) averaged over monthly periods should not exceed 0.01 $\mu g/m^3$. (No absolute limit for peak periods was stipulated.)

Annual review of these standards was made by the AEC for 7 years subsequent to their adoption in 1949, with no cause for revision noted.

The value of 2 $\mu g/m^3$, as the average concentration to which persons may safely be exposed over the course of a normal workday, was adopted by the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists in 1959. In 1956, the American Industrial Hygiene Association published a Hygienic Guide on Beryllium which adopted the values set forth by the Atomic Energy Commission.

The New York State Air Pollution Control Board[6] has set a limit of 0.01 $\mu g/m^3$ for a 24-hour average as the ambient air quality objective for all its regions. This same value was adopted by the Pennsylvania Air Pollution Commission[3] in 1967, based on an extensive study conducted by the Pennsylvania Department of Health in an area surrounding a beryllium plant.

In 1966, the Advisory Center on Toxicology of the National Academy of Science—National Research Council reported[2] on a

study of the toxicity and hazards of beryllium conducted in behalf of the U.S. Public Health Service and the Department of Defense. This report recommended tentative Air Quality Criteria. (These values were considered by the National Academy of Science—National Research Council to be interim criteria, pending the results of additional research into toxicities of beryllium compounds.) These tentative criteria are as follows:

(1) For continuous exposure, a level of 0.01 $\mu g/m^3$, averaged over a 30-day period, applies.

(2) For intermittent exposure to beryllium compounds arising from rocket motor firings, the following limits apply:

A. Soluble beryllium compounds: 75 $\mu g\text{-}min/m^3$ within the limits of 10-60 minutes, accumulated during any two consecutive weeks.

B. Beryllium oxide comparable to a product calcined at temperatures around 400°C: 75 $\mu g\text{-}min/m^3$ within the limits of 10-60 minutes accumulated during any two consecutive weeks.

C. Beryllium oxide comparable to a product calcined at temperatures in excess of 1,600°C: 1,500 $\mu g\text{-}min/m^3$ within the limits of 10-60 minutes accumulated during any two consecutive weeks.

The report continues, "In applying the above criteria

for intermittent exposure to firing of a rocket motor, it will be necessary to consider simultaneously the concentration of soluble beryllium compounds, the 'low-fired' beryllium oxide and the 'high-fired' beryllium oxide, and adjust the limits accordingly. For example, if the rocket effluent is composed of acid-soluble beryllium (36 percent HCl diluted 1:1) in amounts greater than 1 percent but less than 5 percent, the 'high-fired' limit of 1,500 $\mu g\text{-}min/m^3$ should be reduced by a factor of two; if greater than 5 percent, the limit for 'low-fired' beryllium should be used."

It must be emphasized that these proposed criteria are tentative recommendations, subject to further research into the toxicities of beryllium compounds. These criteria have been seriously questioned by recognized authorities in the field of beryllium and its toxic effects.[25]

In the Soviet Union, hygienic regulations have set the maximum concentration of beryllium in production facilities at 1 $\mu g/m^3$.[33] Mil'nikov[40] recommends a value of 1 to 2 $\mu g/m^3$ for beryllium acetate, and a value of 10 to 20 $\mu g/m^3$ for finely dispersed beryllium oxide, as limiting concentrations in factory facilities for a 6-hour day.

Imposition of the AEC limits of exposure has undoubtedly resulted in effective control of beryllium disease in the United States. However, many instances are noted in the literature which suggest that these levels may be overly conservative,[11,32,53] and that additional research is indicated

concerning the toxicity of individual beryllium compounds, maximum tolerance levels, and the severity of exposure as a result of length of time exposed.

3. SOURCES

3.1 Natural Occurrence

Beryllium (Be), one of the lightest of metals, is widely distributed geographically, but exists in relatively small quantities, comprising less than 0.006 percent of the earth's crust. In concentrated form, it is found in relatively few minerals, and these are basically compounds of beryllium oxide. The most important such minerals are as follows, listed alphabetically:[34]

Beryl	$3BeO \cdot Al_2O_3 \cdot 6SiO_2$
Beryllonite	$NaBePO_4$
Bertrandite	$Be_4Si_2O_7(OH)_2$
Bromellite	BeO
Chrysoberyl	$Be(AlO_2)_2$
Euclase	$BeHAlSiO_5$
Hambergite	$Be_2(OH)BO_3$
Helvite	$Mn_4Be_2Si_3O_{12}S$
Herderite	$CaBePO_4(OH,F)$
Leucophanite	$(Ca,Na)_2BeSi_2(O,OH,F)$
Phenacite	Be_2SiO_4

Physically, beryllium has a very high strength-to-weight ratio, great stiffness (exceeding other metals, including steels), high melting point (1,285°C), and valuable nuclear properties. Pure beryllium is quite stable and tarnishes slightly at room temperatures in dry air and in pure oxygen.

When finely ground it burns with brilliant scintillations. Its physical properties are tabulated in Table 14, Appendix.

3.2 Production Sources

The processes of extracting, refining, and machining of beryllium-containing material produce considerable quantities of airborne beryllium, and are considered the principal sources of beryllium illness and poisoning. Dusts are created in crushing, grinding, and cutting operations. Fumes result from condensation of vapors created during melting, pouring, or welding processes; the fumes consist primarily of the oxide. Mists of soluble beryllium compounds occur from such operations as wet grinding and polishing; use of coolant liquids reduces the atmospheric concentration, but the contaminated liquid then becomes a hazard.

The open-pit mining of ores, the recovery of beryllium as the oxide, and the reduction of the oxide have not been accompanied by toxic manifestations.[21,24,62] It is only where finely divided metal, oxide, or compounds have been processed that serious toxic effects have been experienced among beryllium workers and others working or residing near plants from which beryllium-containing dust or fumes have been ejected.[7,41]

3.2.1 Mining

Beryl has been the only commercial source of beryllium, and as there is extremely little commercial-grade beryl produced in the United States, all beryl for consumption has been imported.[41] While beryl is widely distributed, it has never

been found in quantities sufficient to permit the mining of this material for itself. Therefore, it is produced only as a by-product of the mining of other minerals, such as mica, feldspar, etc. There are no known cases of human berylliosis caused by inhalation of beryl dust, and mining is not considered a significant factor in beryllium air pollution.[21,24,62]

In the United States, the minerals bertrandite and phenacite are considered possible sources of beryllium, despite their low grade (0.1-3 percent). These minerals may possibly constitute a hazard to health.[53]

3.2.2 Extraction and Refining

Beryl is converted to beryllium oxide by one of the following processes:[7]

(1) Joy-Windecker Process—Clifton Product, Inc.

In this process the ore is fused with soda ash and the product treated with strong sulfuric acid to obtain silica in insoluble and filterable form, and aluminum and beryllium in the form of water-soluble sulfates.

The major portion of the aluminum sulfate is first separated from the beryllium sulfate by adding ammonium sulfate to the mixed sulfates. The ammonium alum crystals formed by this addition are separated, leaving a filtrate of beryllium sulfate contaminated mainly with aluminum and iron. The iron is oxidized to the ferric state, and the filtrate is diluted and treated with sodium carbonate to a pH of about 5.5. The

precipitate, containing about 95 percent of the aluminum and iron and about 10 percent of the beryllium present, is filtered off and separated. This crude hydroxide is then redissolved in sulfuric acid, the beryllium sulfate solution produced is diluted, and the pH of the solution is adjusted with ammonia to 5.5 to 5.7 to precipitate the remaining aluminum and iron as hydroxides. After separation of these impurities, the filtrate is completely precipitated with ammonia. The pure beryllium hydroxide formed is separated by filtration, washed, dried, and ignited to beryllium oxide of high purity.

(2) Fluoride Process (Copaux-Kawecki)—The Beryllium Corporation

The ore is first ground in a ball mill, either wet or dry, until the particles are about 0.07 mm in diameter. It is then mixed with sodium ferric fluoride and made into wet briquets, which are heated for about an hour at 750°C. The sodium ferric fluoride reacts selectively at 750°C with the beryllium oxide in the beryl ore, forming sodium beryllium fluoride and ferric oxide according to the equation:

$$3BeO \cdot Al_2O_3 \cdot 6SiO_2 + 2Na_3FeF_6 =$$
$$3Na_2BeF_4 + Fe_2O_3 + Al_2O_3 + 6SiO_2$$

(3) Sulfate Process (Sawyer-Kjellgren Process)—Brush Beryllium Co.

The beryl ore is first completely melted at approximately 1,625°C and then quenched in cold water. The melted beryl is

poured into water to obtain a frit, which is ground in a ball mill. This renders the ore susceptible to attack by sulfuric acid at 275°C. The main reactions involved are:

$$3BeO \cdot Al_2O_3 \cdot 6SiO_2 + 6H_2SO_4 =$$
$$3BeSO_4 + Al_2(SO_4)_3 + 6SiO_2 + 6H_2O$$

$$3BeSO_4 + Al_2(SO_4) + (NH_4)_2SO_4 (excess) =$$
$$3BeSO_4 + 2AlNH_4(SO_4)_2 + (NH_4)_2SO_4$$

$$6BeSO_4 = 6BeO + 6SO_2 + 3O_2$$

Beryllium is produced as solid-lump metal by reducing an excess amount of beryllium fluoride in solid form with magnesium metal charged in a graphite-lined furnace at a crucible temperature of about 900°C. A slag of magnesium fluoride and beryllium fluoride is produced. Solid beryllium metal is recovered after the reaction is completed by raising the temperature to 1,300°C., somewhat above the melting point of beryllium. The molten beryllium, which floats on top of the molten slag, is collected into a pool and then quickly solidified into a floating cake of solid beryllium.

This process is based on the following reactions:

$$BeO + 2NH_4F \cdot HF = (NH_4)_2BeF_4 + H_2O$$

$$(NH_4)_2BeF_4 = 2NH_4F + BeF_2$$

Eisenbud *et al.*[16] estimated in a careful engineering study that one extraction plant discharged 5 kg of beryllium per day.

Hazardous concentrations can be expected whenever

certain beryllium compounds are heated even to moderate temperatures. The following table (Table 4), from Tepper et al.[57] indicates the variation in emissions caused by increasing the temperature of different compounds.

TABLE 4

THEORETICAL AIR CONCENTRATIONS OF BERYLLIUM BASED ON VAPOR PRESSURES OF MATERIALS[57]

Compound	Temperature (°C)	Beryllium Concentration (μg/m³)
Beryllium	885	2
	1,060	200
Beryllium oxide	1,710	2
	1,990	200
Beryllium oxide and water vapor	1,400	0.68×10^6
Beryllium fluoride	420	2
	525	200
Beryllium chloride, iodide, bromide	108	2
	158	200
Beryllium borohydride	20	4.35×10^6

Prior to recognition of the hazardous nature of this metal, the dusts, mists, and fumes resulting from industrial operations were permitted to pass into the atmosphere without control. In 1947-48, a period in which controls were just coming into general use, the AEC conducted a survey of occupational exposures within beryllium plants. The following tables present data on pollution concentrations in various phases of the industry:

TABLE 5

BERYLLIUM OXIDE PRODUCTION
LORAIN, OHIO[63]

Process	Air Pollution Data[a]		
	G.A.[b]	B.Z.[c]	G.A.-B.Z.[d]
Ore reduction	440; 212; 12	212; 1,350 380; 267 235; 102 318	
Sulfating (hot mill)	173; 28; 96	230; 20	150; 600
Sludge repulp. (wet process)	2	176	
$BeSO_4$ crystallizing (wet process)	22; 17; 2 15; 11; 24 1.4; 9; 42	45,200; 270 16.2; 31 247; 32; 18 12; 14; 55 28	1,135; 27 420; 18; 19 140
$BeSO_4$ calcining	6,450	83,000 76,000; 2,100 3,500	

[a]Air pollution data recorded in $\mu g/m^3$, air samplings taken between 10-23-47 and 2-26-48. Total: 50, listed in chronological sequence.

[b]GA = General air within the plant.

[c]BZ = Breathing zone of a given worker on a specific operation.

[d]GA-BZ = Transitionary exposure to both breathing zone and general air conditions.

Table 6 gives data for 1947-48 on the environmental concentrations to which workers were exposed in the fluoride process of producing metallic beryllium from the beryllium oxide:

TABLE 6

BERYLLIUM METAL PRODUCTION
LORAIN, OHIO[63]

Process	Air Pollution Data		
	G.A.[b]	B.Z.[b]	G.A.-B.Z.[b]
$(NH_4)_2BeF_4$ (wet process)	180; 770 19,000; 200 16; 6.2		
$(NH_4)_2BeF_4$ decomp.	36; 746; 48 200; 97; 44 33; 110; 35 110; 35; 100 8; 10; 7; 21	49; 184; 2,118 1,765; 8; 16 380	23
BeF_2 crushing		2,295; 550	
BeF_2 reduction		187; 146	
Melts crushing	8; 23; 7; 76	2,470; 6,000	4,950
Pebble: leaching, flotation (wet), drying, sorting, screening (dry)	28.2; 177 4.8	1,320; 1,128 9,010; 883 27; 196	

[a]Air pollution data recorded in $\mu g/m^3$, air samplings taken between 10-23-47 and 2-26-48. Total: 48, listed in chronological order.

[b]See footnotes for Table 5.

Table 7 presents similar data for the powdering, sintering, and milling of beryllium:[62]

TABLE 7

BERYLLIUM METAL POWDERING, SINTERING, MACHINING LORAIN, OHIO, AND CLEVELAND, OHIO[63]

Process	Air Pollution Data[a]		
	G.A.[b]	B.Z.[b]	G.A.-B.Z.[b]
Rolling Be chips	1,461; 324	35,890 11,914	
Bottling chips	41	8,687	
Attrition mill		1,227; 1,277	
Powder drying	51		
Sintering	32.7; 184.6 16; 7; 28; 37 65		
Machining		461; 70; 35 45; 38; 32	

[a]Air pollution data recorded in $\mu g/m^3$, air samplings taken between 10-23-47 and 2-26-48. Total: 23, listed in chronological order.

[b]See footnotes for Table 5.

The major producers of beryllium in the United States are:

The Beryllium Corporation, Reading and Hazelton, Pa.
Brush Beryllium Company, Elmore, Ohio

The world and U.S. production of beryl for the years 1957 to 1966 are presented in Table 8.

TABLE 8

PRODUCTION OF BERYL[53]

	Short Tons					
	1957-61	1962	1963	1964	1965	1966
World	11,080	11,000	7,700	5,200	4,900	3,600
United States	U[a]	978	751	W[b]	W[b]	W[b]

[a]U = Unknown.

[b]W = Withdrawn: company confidential.

Research into new uses for beryllium, plus the impetus of the aircraft and space industries' interest in this metal, indicates increasing demand for beryllium production in the future.

3.3 Product Sources

Beryllium has found a number of important uses in industry. These uses are of significance to air pollution mainly during the initial processing period; however, in certain instances hazards to health continue to exist after the production of the particular product. The wide variety of uses to which beryllium is being put may cause unintentional worker exposure to levels of beryllium which may pose a threat to health. This is particularly true of small machine shops and foundries, which tend to have inadequate industrial health practices. An additional hazard is the possibility of neighborhood contamination arising from small plants having

inadequate pollution control devices.

3.3.1 Beryllium-Copper Alloys

A large part of the beryllium produced is used as a hardening agent in alloys, the most common of which is beryllium copper. Depending upon the beryllium content, the alloy can be high strength (high beryllium content) or high-conductivity (low beryllium content). This alloy is formed by the direct reduction of beryllium oxide with carbon in the presence of copper, resulting in a 2 to 4 percent beryllium alloy. Dusts and fumes of beryllium oxide can be emitted in the alloying process and in the reduction process to refine scrap alloy. The proportion of beryllium to the basic metal is not a reliable index of the degree of hazard attending the manufacture of the alloy. Tepper et al.[57] reported on cases of beryllium poisoning occurring from production of copper alloy of 2.75 percent beryllium, during machining and surface grinding, when no control procedures were used. Air sampling revealed 0.39 to 0.78 $\mu g/m^3$ during melting and pouring, 3.55 to 21.20 $\mu g/m^3$ during drossing and casting, and 87 to 144 $\mu g/m^3$ during dry surface grinding.

Table 9 indicates the concentrations of beryllium in the air at a number of locations within a beryllium-copper alloy plant,[12] in the year 1960, as observed by the Kettering Laboratory, University of Cincinnati, Ohio.

3.3.2 Fluorescent Tubes

Beryllium oxide is no longer used in the manufacture

TABLE 9

MEAN, MEDIAN, AND RANGE VALUES OF BERYLLIUM CONCENTRATIONS (2-HOUR AVERAGES)[12]

Location	Hours	Concentration in $\mu g/m^3$ Median	Average	Range
Oxide area	92	72.5	149.4	0.4 -1,050.0
Arc furnace area	92	50.0	87.6	22.1 -502.0
Mixing area	92	14.4	21.6	0.03-452.0
Cropping area	92	33.6	52.8	14.0 -399.0
Casting area	86	14.6	39.8	0.2 -535.0
Fischer furnace area	91	28.8	40.8	0.2 -340.0
Oliver saw area	90	21.1	25.6	<2.5 -92.5
All areas	635	28.4	60.3	0.3 -1,050

of fluorescent light tubes. However, much of the history of beryllium disease, and the controls upon the beryllium industry, stem from this usage in the 1940's, and mention should therefore be made of this source of emissions. In the manufacturing process, tube phosphors were prepared using beryllium oxides (ranging from 4 to 13 percent), zinc, magnesium, and manganese, fired at a high temperature. Dusts and phosphor powders were present in high concentrations, but largely unmeasured. The prevalence-of-disease rate ran approximately 3 percent of the population at risk.[57] It was significantly higher in the plant using 13 percent beryllium oxide than in the plants using 4 percent beryllium oxide. In 1949, the use of beryllium

oxide in fluorescent tubes was discontinued.

3.3.3 Rocket Fuels

Finely powdered metallic beryllium may be used as an additive in rocket fuels, to provide increased performance characteristics.[12] Three separate air pollution hazards occur from this usage: handling of the beryllium metal prior to launch; exhaust fumes resulting from firing; and the possible dangers from accidental explosions or fires. Exhaust fumes are predominantly beryllium oxide, with other compounds present depending upon the chemical formulation of the propellant charge. For example, one analysis of rocket exhaust products revealed that 50 percent of the total beryllium was beryllium oxide, 40 percent beryllium fluoride, and the remainder was mainly beryllium chloride.[47]

Analyses of residues from the burning of rocket propellants containing beryllium additives have been conducted by the Hercules Powder Company and the Kettering Laboratory.[12] These analyses revealed that a number of compounds of beryllium, including the chloride and the oxide, as well as metallic beryllium, were present in the residue from the propellants. Approximately 70 percent of the total metallic beryllium used in the propellant was recovered in the residue, indicating that 30 percent presumably escaped into the atmosphere.

The handling, storage, and use of beryllium metal as a rocket fuel is obviously a localized problem.

3.3.4 Coals

Beryllium is a constituent of most coals, in varying concentrations. The average contents in coal of the three major coal-producing areas of the United States are:[1]

Area	Average	Minimum	Maximum
Northern Great Plains	1.5 ppm	<0.1	9.1
Eastern Interior	2.5 ppm	0.5	12.0
Appalachian Region	2.5 ppm	0.1	31.0

Despite these low concentrations, the amount of beryllium added to the atmosphere by the burning of coal may be significant, in view of the quantity of coal consumed in the United States.

3.4 Other Sources

The laundering of garments used by beryllium workers can be a significant source of beryllium in the local air. Eisenbud *et al.*[16] reported that home laundering of clothes contaminated with beryllium dusts could result in inhalation of 17 μg of beryllium per day. Tepper *et al.*[57] mention a study of garments used by workers performing cylindrical grinding of beryllium parts: aprons contaminated by spattering of coolant and by the operators' hands produced a concentration of 7.7 $\mu g/m^3$ of beryllium when shaken in a dust chamber.

As noted in Section 2, some "neighborhood" cases of beryllium poisoning resulted from home laundry of workers' garments.

3.5 Environmental Air Concentrations

Beryllium is known to be present in the atmosphere, and measurements have been made of its concentrations. Beryllium is now included in routine measurements of atmospheric constituents from the National Air Sampling Networks. Air Quality Data figures[4] of average and maximum concentrations reveal that the daily average atmospheric concentration of beryllium for 100 stations in the United States is less than 0.0005 $\mu g/m^3$, and the maximum value recorded for the 1964-65 period is 0.008 $\mu g/m^3$. Tabular data for these 100 stations are listed in Table 15, Appendix. Chambers et al.[10] analyzed air samples collected from over 30 metropolitan areas, and found a maximum value of 0.003 $\mu g/m^3$. The variation of average atmospheric beryllium concentrations from urban areas (over 2 million population) to small cities to rural areas is demonstrated by the following data from Chambers et al.[10] in Table 10. The authors acknowledge the limitations of this work, including the methods used, the selection of the locations, extent of coverage, and the inherent defects in analysis of data based only on particulate samples; however, despite these limitations, their work provides useful comparative data.

An air pollution study of atmospheric concentrations of beryllium in the vicinity of a large beryllium plant near Reading, Pa., was conducted in 1958.[56] The average concentration within one-half mile of the plant was 0.0281 $\mu g/m^3$; concentrations closer to the plant reached 0.0827 $\mu g/m^3$.

TABLE 10

AVERAGE BERYLLIUM CONCENTRATIONS FOR SELECTED AREAS[10]

Area	Concentration ($\mu g/m^3$)
Cities over 2,000,000	
Los Angeles	0.0001
Detroit	0.0004
Philadelphia	0.0005
Chicago	0.0002
New York	0.0003
Cities between 500,000-2,000,000	
Cincinnati	0.0002
Kansas City	0.0003
Portland (Oreg.)	0.0003
Atlanta	0.0002
Houston	0.0002
San Francisco	0.0001
Minneapolis	0.0002
Rural (Suburban)	
Boonsboro (Md.)	0.0001
Salt Lake City	0.0001
Atlanta	0.0002
Cincinnati	0.0001
Portland (Oreg.)	0.0001

4. ABATEMENT

Efforts to reduce concentrations of beryllium in the atmosphere have generally centered on the problem of reducing industrial concentrations of beryllium, where exceptional success has been achieved. Control activity has mainly surrounded significant beryllium plants, where concentrations of beryllium dust, produced within the plant, are discharged to the outside atmosphere through intent or by accident. Recognition of beryllium as a cause of industrial disease in the early 1940's has led to sweeping changes in manufacturing processes and controls designed to reduce the concentration of atmospheric beryllium, both in-plant and out-of-plant. Conventional air-cleaning devices have been used. For wet chemical processes, scrubbers, venturi scrubbers, packed towers, organic wet collectors, and wet cyclones have been found to be effective. For dry processes, conventional bag collectors, reverse-jet bag collectors, electrostatic precipitators, cyclones, and unit filters have been used. Table 11 contains a listing by Silverman[49] of air-cleaning devices and their expected efficiency.

The effectiveness of abatement activities can be appreciated by comparing present-day industrial exposure concentrations with concentrations existing prior to recognition of the significance of beryllium air pollution to industrial health. Breslin[9] states that current practices limit employees'

TABLE 11

CLEANERS FOR BERYLLIUM HANDLING OPERATIONS[49]

Operation or Process Phase	Type Cleaner	Expected Efficiency by Weight (Percent)
Ore handling, crushing, etc.	Reverse-jet or shaking bag filter	99
Sinter furnace	Wet cell or spray scrubber	80
Leaching and hydroxide filter	Wet cell or spray scrubber	80
Sodium fluoride handling	Wet cell or spray scrubber	80
Beryllium hydroxide, dry	Reverse-jet or shaking bag filter	99
Beryllium hydroxide drier and calciner	Wet spray for cooling, then reverse-jet or shaking bag filter	99
Beryllium fluoride mixer	Wet cell or spray tower	80
Beryllium fluoride furnace	Venturi or orifice scrubber or packed tower and wet Cottrell unit	95
Reduction furnace	Venturi or orifice scrubber or packed tower and wet Cottrell unit	95
Ball mill	Wet cell washer	80
Machinery, powder, metals handling	Small cyclone units plus bag filter with asbestos filter aid	99.9
Welding, heat-treating	Bag filter with filter aid and dilution air to bring temperature to 180°F	99.9
Miscellaneous laboratory hoods	Roughing filter plus high-efficiency AEC-type filter	99.95

exposure to beryllium dust to about 2 $\mu g/m^3$ or less, with out-of-plant neighborhood concentrations limited to 0.01 $\mu g/m^3$. By comparison, University of Rochester[35] samples of beryllium concentration in a Cleveland factory in 1946 revealed concentrations as high as 4,710 $\mu g/m^3$.

Another form of control is exemplified by discontinuance of the use of beryllium in fluorescent lamp tubes in 1949; this action eliminated an industrial exposure to beryllium which had been responsible for approximately one-third of all beryllium respiratory illnesses.[17]

5. ECONOMICS

No losses resulting from damage to plants or materials have been recorded from beryllium air pollution. It is difficult to assess the economic costs of loss of life or illnesses resulting from beryllium disease, and no statistical data have been found on this subject. However, court cases are now pending in Pennsylvania that are based on impairment of health resulting from beryllium exposure.

Schulte and Hyatt[62] reported one particularly detailed study on the economics of industrial hygiene control in a beryllium machinery plant in the Los Alamos Scientific Laboratory (New Mexico) in 1958. The cost figures produced by this study are as follows:

Item	Cost/Year
Construction of shop area to incorporate control facilities (1,000 sq. ft., at $22.00/sq.ft. amortized over 20 years)	$ 1,100.00
Ventilation system ($12,000 depreciated over 15 years at annual cost of $800)	800.00
Clothing for four workers (coveralls, shirts, shorts, socks, caps, and shoe covers)	274.00
Additional work-time required by hygienic procedures necessary for beryllium control	8,000.00
Air sampling equipment (amortized over 5 years). .	160.00
Air sampling operations	250.00
Chemical analysis at $5.00/sample, 344 samples . .	1,720.00
Laundering of shop clothing	1,000.00

(continued)

Item	Cost/Year
Janitorial (no extra costs)	$
Filter changing and ventilation maintenance . . .	112.00
Trash disposal	150.00
Medical examinations ($25.00/man)	100.00
Total costs	$13,666.00

As the average cost of machine shop operation for the four-man shop is $70,000 per year, the added costs for hazard control run approximately 20 percent.

According to Tepper,[57] in many instances the recovery of valuable materials will significantly offset the costs of abatement of beryllium air pollution.

Data on the production and consumption of beryllium are presented in Section 3.

6. METHODS OF ANALYSIS

6.1 Sampling Methods

The most common method of sampling beryllium concentrations in air employs a high-volume sampler which draws air to be analyzed through a filter for the appropriate sampling period.[13] The Hi-Vol sampler—which samples at a rate of 1.5 to 1.7 m^3/min when operated 24 hours—will provide an adequate sample for the measurement of beryllium in ambient air in concentrations as low as 0.002 $\mu g/m^3$. Cellulose-fiber filter paper with very low ash content is widely used; cellulose ester and fiberglass papers are also used, and are particularly suitable for use when collecting contaminants are of very small size.

6.2 Quantitative Methods

Atmospheric beryllium may be analyzed by colorimetric, fluorimetric, or spectrographic procedures.[11] These methods are accurate and sensitive but do not discriminate between the various compounds of beryllium.

6.2.1 Morin Fluorescent Method

The morin method is suitable for measuring quantities of beryllium in the air surrounding beryllium plants; its lower limit of detection is approximately 0.01 µg of beryllium.[11] In this procedure, the beryllium is removed from the filter, processed to remove interfering elements and to form an alkaline solution, and fluorescent morin solution ($C_{15}H_{10}O_7 \cdot H_2O$)

is added. Beryllium acts to reduce the fluorescent characteristics of the morin solution, as a direct function of the quantity of beryllium present. Measurement of the fluorescence by fluorimeter against standard samples indicates the quantity of beryllium. (Note: Reference 31 states that the procedure is sensitive to 0.00002 µg/ml of the solution.

6.2.2 Colorimetric Method

This method, commonly called the Zenia method, is suitable for large concentrations from which at least 2 µg of beryllium can be obtained, and is used in monitoring the atmospheres of industrial plants. The procedure involves removal from the filter paper, removal of interfering elements, and addition of the Zenia (p-nitrobenzeneazoorcinol) solution. Zenia solution added to a beryllium solution forms a reddish-brown solution, whose absorption of light is a function of its beryllium content. Measurements can be made subjectively, to give a quick spot-check,[15] or by means of a spectrophotometer. Sensitivity to 0.5 µg/ml of solution is claimed.[31]

6.2.3 Spectrographic Method[11]

This spectrographic method is considered suitable for measuring concentrations in the general atmosphere, as it permits detection of trace quantities down to 0.003 µg.[11] Also, automatic detectors are available that are capable of handling large numbers of samples rapidly and accurately. This method adds a thallium standard to the beryllium-zenia solution

produced in the colorimetric method, concentrates the solution, subjects it to an electric arc, and photographs its spectra. The ratio of the intensity of the beryllium line at 2348.6Å to that of the thallium line at 2379.7Å is determined, giving a measure of the concentration of beryllium.

McCloskey[38] reported on a variation of this method using aluminon reagent in place of the Zenia solution that was said to be more sensitive than the normal spectrographic procedure.

6.2.4 Other Methods

Another method for automatic monitoring of beryllium in air, under development by the U.S. Air Force, was reported on in 1963 by Braman[8] of the ITT Research Institute. This device attempted to use the nuclear reaction of beryllium for the detection of beryllium. Design goals called for an alarm signal at 25, 2, and 0.01 $\mu g/m^3$ of beryllium over sampling periods of 30 seconds, 1 hour, and 24 hours respectively.

Emission spectroscopy has been used by the National Air Pollution Control Administration for beryllium analysis of samples from the National Air Sampling Network.[58] The samples are ashed and extracted to eliminate interfering elements. The minimum detectable beryllium concentration by emission spectroscopy is 0.0008 $\mu g/m^3$ for urban samples and 0.00016 $\mu g/m^3$ for nonurban samples. The different sensitivities result from the different extraction procedures required

for urban samples.[7]

The National Air Pollution Control Administration uses atomic absorption to supplement analyses obtained by emission spectroscopy. The method has a minimum detectable limit of 0.01 $\mu g/m^3$ based on a 2,000 m^3 air sample.[58]

7. SUMMARY AND CONCLUSIONS

Inhalation of beryllium or its compounds is highly toxic to humans and animals, producing body-wide systemic disease commonly known as beryllium disease. Both acute and chronic manifestations of the disease are known. The effects of beryllium intoxication can be mild, moderate, or severe, and can prove fatal, depending on the duration and intensity of exposure.

Acute beryllium disease is manifested by a chemical pneumonitis ranging from transient pharyngitis or tracheobronchitis to severe pulmonary reaction. As of June 1966, 215 acute cases had been recorded in the Beryllium Case Registry.

Chronic beryllium disease generally occurs as lesions in the lung, producing serious respiratory damage and even death. However, every organ system may be involved in response to beryllium exposure, except for the organs in the pelvic area. The chronic form is characterized by a delay in onset of disease, which may occur weeks or even years after exposure. In June 1966, 498 chronic cases had been recorded, plus 47 acute-to-chronic cases.

Of the total 760 cases recorded in the Beryllium Case Registry, 210 fatalities, or 27.5 percent, had occurred by June 1966.

Cancer has been produced experimentally in animals,

and 20 cases of cancer have been found (as of 1966) in humans afflicted with beryllium disease. However, insufficient information exists at this time to causally relate beryllium poisoning to development of cancer in humans.

Beryllium and its compounds can produce dermatitis, conjunctivities, and other contact effects; however, these manifestations are rare.

There is some evidence that beryllium in soils is toxic to plant life; no evidence was found on the effects of atmospheric beryllium on plants or on materials.

The major potential sources of beryllium in the atmosphere are industrial. The processes of extraction, refining, machining, and alloying of the metal produce toxic quantities of beryllium, beryllium oxide, and beryllium chloride, which if allowed to escape into the atmosphere would cause serious contamination. Recognition of the serious hazards to health from these sources has led to adaptation of control procedures minimizing this potential. However, beryllium in limited quantity is emitted from these industrial processes, and danger also exists from accidental discharges. One major source of beryllium contamination—the use of beryllium in fluorescent light tubes—was discontinued in 1949. Other sources could be the use of metallic beryllium in rocket fuels, and the combustion of coals. Rocket fuels could present a hazard in the handling and storage of the powdered metallic beryllium

used as an additive in the fuels. Also, the exhaust fumes, which contain oxidized beryllium as well as other compounds of beryllium, would be of significance in local soil and air pollution if not contained. As beryllium is a normal constituent (above 2 ppm) of coals, the combustion of coal may add a significant quantity of beryllium to the atmosphere.

Measurements are made of the beryllium concentration at 100 stations in the United States. The average 24-hour concentration is less than 0.0005 $\mu g/m^3$; the maximum value recorded during the 1964-65 period was 0.008 $\mu g/m^3$.

Abatement measures have been implemented industry-wide, with a very high degree of success. Conventional air-cleaning procedures have been employed, including the use of electrostatic precipitators, bag houses, scrubbers, etc. These procedures have enabled the beryllium industry to meet the industrial hygiene standards established for beryllium.

Data on the economic losses resulting from beryllium air pollution are not available. Court cases are pending in the State of Pennsylvania, however, which may provide data on the economic values of impairment to health resulting from exposure to beryllium. Only one analysis of the costs for abatement was found. This study indicated that the added costs for control amounted, in 1952, to approximately 20 percent of the normal cost of operation for the particular plant analyzed.

Methods of analyzing beryllium in the atmosphere are available, and are adequate for normal industrial processes. The most common methods are the Zenia method, the morin fluorescent method, and the spectrophotometric method. The Zenia method is relatively simple, works well with high concentrations, and provides sensitivity on the order of 0.5 $\mu g/m^3$. It can also be used subjectively, to provide a quick spot-check for the presence of beryllium materials. The morin fluorescent method provides a higher sensitivity range (0.01 $\mu g/m^3$) and is suitable for monitoring out-of-plant concentrations in the vicinity of beryllium processing plants if a large enough volume of air is sampled. The spectrographic process gives even higher sensitivities (0.003 $\mu g/m^3$) and is suitable for monitoring concentrations in the general atmosphere. However, none of the currently available procedures provides for discrimination between the various compounds of beryllium, or differentiation between the "low-fired" (highly-toxic) and the "high-fired" (less toxic) forms of beryllium oxide.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Further research into the pathogenesis of beryllium disease, with particular emphasis upon the effects of protracted exposures to low concentrations.

(2) Further research into the carcinogenicity of beryllium compounds.

(3) Analysis of the contribution of coal combustion to beryllium pollution of the atmosphere.

(4) Development of procedures for analysis of different compounds of beryllium present in the atmosphere.

(5) Development of improved methods for characterization of combustion products of rocket fuels containing beryllium compounds.

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APPENDIX

APPENDIX

TABLE 12. PROPERTIES, TOXICITY, AND USES OF BERYLLIUM AND SOME BERYLLIUM COMPOUNDS[38]

Compound	Properties	Toxicity	Uses
Beryllium Be	mp 1,284°- 1,300°C bp 2,970°C gray metal	Death may result from short exposure to incredibly low concentrations of the element and its salts. Concomitant exposure to acid fumes (hydrofluoric) may increase toxic effects. Contact dermatitis, chemical conjunctivitis, corneal burns, nonhealing ulceration at site of injury, subcutaneous nodules may occur following exposure. Acute: pneumonitis may result from single exposure to beryllium and occasionally is fatal. Chronic: pulmonary granulomatous disease may appear in 3 months to 15 years, often after short exposure to low concentration. Uncertainty as to complete recovery. Death rate about 25%	As source of neutrons when bombarded with alpha particles, according to the equation $^{9}_{4}Be + ^{4}_{2}He \rightarrow ^{12}_{6}C + ^{1}_{0}n$. This yields about 30 neutrons per million alpha particles. Also as neutron reflector and neutron moderator in nuclear reactors. In beryllium copper and beryllium aluminum alloys (by direct reduction of beryllium oxide with carbon in the presence of Cu or Al). In radio tube parts, television tube phosphors, fluorescent tubes
Beryllium acetate $C_4H_6BeO_4$	Decomposes 60-100°C Crystals	See beryllium	
Beryllium acetate, basic $Be_4O(OCOCH_3)_6$	mp 285-286°C bp 330-331°C	See beryllium	

(continued)

TABLE 12. PROPERTIES, TOXICITY, AND USES OF BERYLLIUM AND SOME BERYLLIUM COMPOUNDS (Continued)

Compounds	Properties	Toxicity	Uses
Beryllium acetylacetonate $C_{10}H_{14}BeO_4$	Monoclinic crystals. mp 108°C bp 270°C	See beryllium	
Beryllium borohydride $Be(BH_4)_2$	Spontaneously flammable Sublimes at 91.3°C Decomposes above 123°C	See beryllium	
Beryllium bromide $BeBr_2$	Crystals mp 488°C Sublimes at 473°C bp 520°C	See beryllium	
Beryllium carbide Be_2C	Decomposes above 2,100°C Brick-red or yellow-red octahedra	See beryllium	
Beryllium chloride $BeCl_2$	White to faintly-yellow crystals mp 405°C bp 488°C		In manufacture of beryllium. Anhydrous form used as acid catalyst in organic reactions similar to $AlCl_3$

(continued)

APPENDIX

TABLE 12. PROPERTIES, TOXICITY, AND USES OF BERYLLIUM AND SOME BERYLLIUM COMPOUNDS (Continued)

Compounds	Properties	Toxicity	Uses
Beryllium fluoride BeF_2	Glassy hygroscopic mass	See beryllium	In manufacture of Be and Be alloys, manufacture of glass; in nuclear reactors
Beryllium formate $Be(OOCH)_2$	Powder, decomposes above 250°C Sublimes at 320°C	See beryllium	
Beryllium hydride BeH_2	White solid	See beryllium	
Beryllium hydroxide $Be(OH)_2$	Amorphous powder or crystals	See beryllium	In manufacture of beryllium and beryllium oxide
Beryllium iodide BeI_2	Needles mp 480°C bp 488°C	See beryllium	
Beryllium nitrate $Be(NO_3)_2$	White to slightly yellow crystals mp ~60°C	See beryllium	For stiffening mantles in gas and acetylene lamps

(continued)

TABLE 12. PROPERTIES, TOXICITY, AND USES OF BERYLLIUM AND SOME BERYLLIUM COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Beryllium nitride Be_3N_2	White crystals to grayish-white powder mp 2,200 ± 40°C	See beryllium	
Beryllium oxide BeO	Light amorphous powder mp 2,530°C	See beryllium	In manufacture of beryllium oxide ceramics, glass; in nuclear reactor fuels and moderators; as catalyst for organic reactions
Beryllium perchlorate $Be(ClO_4)_2$	Hygroscopic crystals	See beryllium	
Beryllium potassium fluoride K_2BeF_4	Hard masses	See beryllium	
Beryllium potassium sulfate $K_2Be(SO_4)_2$	Brilliant crystals Soluble in water	See beryllium	In chromium and silver plating
Beryllium selenate $BeSeO_4$	Orthorhombic crystals Anhydrous at 300°C	See beryllium	

(continued)

APPENDIX

TABLE 12. PROPERTIES, TOXICITY, AND USES OF BERYLLIUM AND SOME BERYLLIUM COMPOUNDS (Continued)

Compounds	Properties	Toxicity	Uses
Beryllium sodium fluoride $NaBeF_4$	Orthorhomic or monoclinic crystals mp ~350°C	See beryllium	
Beryllium sulfate $BeSO_4$	Crystals at about 100°C Loses $2H_2O$	LD_{50} in mice: 500 µg/kg Human toxicity: see beryllium	

APPENDIX

TABLE 13

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle size μ	Effects
Beryllium fluoride	5 cats, young adults	970 μg/m³ in H_2O	6 hr/day (207 days)	0.61(0.33-0.94)	No deaths; lung damage
	6 cats, young adults	10,000 μg/m³ in H_2O	6 hr/day (3 wk)	0.63(0.52-0.74	No deaths
	14 dogs, young adults	970 μg/m³ in H_2O	6 hr/day (207 days)	0.61(0.33-0.94)	3 deaths; suspected macrocytic anemia; Consolidation, emphysema, and slight edema in lungs; Be tended to accumulate in lungs, pulmonary lymph nodes, liver, skeleton, and bone marrow
	6 dogs, young adults	10,000 μg/m³ in H_2O	6 hr/day (3 wk)		1 death; 3 dogs in moribund condition sacrificed
	6 dogs, young adults; 3 rabbits	2,700 (2,000-2,400 μg/m³ in H_2O	6 hr/day (23 wk)		Decrease in RBC count and Hb levels; increase in mean corpuscular volume consistent with macrocytic anemia
	20 guinea pigs, young adults	10,000 μg/m³ in H_2O	6 hr/day (3 wk)	0.63(0.52-0.74)	7 deaths
	20 mice, young adults	10,000 μg/m³ in H_2O	6 hr/day 5 days/wk (3 wk)	0.63(0.52-0.74)	6 deaths
	10 rabbits, young adults	970 μg/m³ in H_2O	6 hr/day (207 days)	0.61(0.33-0.94)	No deaths; suspected macrocytic anemia; lung damage
		10,000 μg/m³ in H_2O	6 hr/day, (3 wk)	0.63(0.52-0.74)	1 death; suspected macrocytic anemia; lung damage
	120 rats, young adults	970 μg/m³ in H_2O	6 hr/day, 5 days/wk (207 days)	0.61(0.33-0.94)	73 deaths; minimal lung lesions

(continued)

APPENDIX

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium fluoride	40 rats, young and old adults	10,000 μg/m³ in H_2O	6 hr/day, 5 days/wk (3 wk)	0.63(0.52-0.74)	7 deaths; minimal lung lesions
Beryllium oxide	65 rats	89.57 μg/L	1-5 hr/day	0.285(0.11-1.25)	Large amounts of the dust (≤24,000 μg/100 g of Be) in lungs more than a year after exposure; little tendency for Be to be redistributed from lungs to other tissues; fibrous tissue proliferation from 35 days to more than a year after exposure, but no granulomatous inflammation of lungs
	90 rats, Wistar, young adults[a]	10,000 & 82,000 μg/m³ in H_2O[b]	6 hr/day, 5 days/wk (15-40 days)	(0.47-0.59)	Damage in lungs only; dust particles in peribronchial and perivascular tissues, as well as in alveoli and phagocytes; inflammation, edema, and thickening of alveolar walls; bronchial epithelial desquamation and hyperplasia
		83,000 μg/m³ in H_2O[c]	6 hr/day, 5 days/wk (60 days)	1.13	
		(84,000-86,000) μg/m³ in H_2O[d]	6 hr/day, 5 days/wk (10-17.5 days)	<1.0	
		88,000 μg/m³ in H_2O[e]	6 hr/day, 5 days/wk (10 days)	0.71	
Beryllium sulfate	4 cats young adults	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; 20% body weight loss. μg Be/g fresh tissue from 4 sacrificed animals: lung, 0.08; liver, 0.02; kidney, 0.01; spleen, 0.01
	5 cats, young adults	10,000 μg/m³ (430 μg Be) in H_2O	6 hr/day (95 days)	1.5	1 death; no change in body weight

(continued)

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium sulfate	5 cats, young adults	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	4 deaths; 43% body weight loss
	12 dogs	(3,600-4,000) μg μg/m³ in H_2O	6 hr/day (2 mo)		Decrease in RBC count and Hb levels; increase in mean corpuscular volume consistent with macrocytic anemia; spontaneous recovery from anemia after 3.5-4 mo
	5 dogs, young adults	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; 10% body weight loss. μg Be/g fresh tissue from 5 sacrificed animals: lung, 0.6; pulmonary lymph nodes, 0.7; liver 0.01; kidney, 0.003; spleen, 0.01 — Reversible macrocytic anemia after 3-8 wk; significant changes in phospholipid and free cholesterol of whole RBC; tendency to hypoalbuminemia and hyperglobulinemia; acute inflammatory response in lung, with erosion and proliferation of bronchial epithelium
		10,000 μg/m³ (430 μg Be) in H_2O	6 hr/day (95 days)	1.5	No deaths; 11% body weight loss; leukocytosis. μg Be/g fresh tissue from 4 sacrificed animals: lung, 4; pulmonary lymph nodes, 2 liver, 1.8; kidney, 0.8; spleen, 0.004; femur, 0.8
		47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	4 deaths; 4% body weight loss; leukocytosis
	1 goat	47,000 μg/m³ (2,000 μg Be in H_2O	6 hr/day (51 days)	0.96	1 death; no change in body weight

(continued)

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium sulfate	20 guinea pigs (400-600 g)	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; 18% body weight gain
	34 guinea pigs (400-600 g)	10,000 μg/m³ (430 μg Be in H_2O	6 hr/day (95 days)	1.5	2 deaths; 100% body weight gain
	12 guinea pigs (400-600 g)	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	7 deaths; 37% body weight gain
	10 guinea pigs (400-600 g)	100,000 μg/m³ (4,300 μg Be) in H_2O	6 hr/day (14 days)	1.1	3 deaths; 2% body weight loss
	83 hamsters	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; no change in body weight
	10 hamsters	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	5 deaths; 18% body weight loss
		100,000 μg/m³ (4,300 μg Be in H_2O	6 hr/day (14 days)	1.1	2 deaths; 8% body weight loss
	38 mice	47,000 μg/m³ (2,000 μg Be in H_2O	6 hr/day (51 days)	0.96	4 deaths; 6% body weight loss

(continued)

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium sulfate	38 mice	100,000 μg/m³ (4,300 μg Be) in H_2O	6 hr/day (14 days)	1.1	No deaths; 13% body weight loss
	2 monkeys	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; 10% body weight gain. μg Be/g fresh tissue from 2 sacrificed animals: lung, 1.2; pulmonary lymph nodes; 1.3; liver, 0.5; kidney, 0.01; spleen, 0.1
	5 monkeys	10,000 μg/m³ (430 μg Be) in H_2O	6 hr/day (95 days)	1.5	No deaths; 31% body weight loss
	1 monkey	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	1 death; 25% body weight loss
	23 rabbits (2.6-4.0 kg)	950 μg/m³ (40 μg Be) in H_2O	6 hr/day (100 days)	0.25	No deaths; 15% body weight gain. μg Be/g fresh tissue from 5 sacrificed animals; lung, 1.6 pulmonary lymph nodes, 0; liver, 0.004; kidney, 0.003; spleen, 0.01
	24 rabbits (2.6-4.0 kg)	10,000 μg/m³ (430 μg Be) in H_2O	6 hr/day (95 days)	1.5	2 deaths; no change in body weight; leukocytosis
	10 rabbits (2.6-4.0 kg)	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	1 death; 7% body weight gain; leukocytosis
	3 rabbits (2.6-4.0 kg)	100,000 μg/m³ (4,300 μg Be) in H_2O	6 hr/day (14 days)	1.1	No deaths; no change in body weight; leukocytosis

(continued)

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium sulfate	40 rats	4,000 μg/m³ in H_2O	6 hr/day (23 wk)		Decrease in RBC count; increase in mean corpuscular volume consistent with macrocytic anemia
	20 rats (250-280 g)	950 μg/m³ (40 μg Be) in H_2O	6 hr/day, 5 days/wk (100 days)	0.25	No deaths; 20% body weight gain
	15 rats (250-280 g)	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day, 5 days/wk (51 days)	0.96	13 deaths; no change in body weight; leukocytosis
	10 rats (250-280 g)	100,000 μg/m³ (43,000 μg Be) in H_2O	6 hr/day, 5 days/wk (14 days)	1.1	10 deaths; 2% body weight loss; leukocytosis
	136 rats, Wistar & Sherman (140-210 g)	12 μg/ft³ (1 μg Be) 1% in H_2O	8 hr/day, 5½ days/wk (6 mo)		46 deaths. Apparent effect on lung tissue: stimulation of epithelial cell proliferation without provoking a connective tissue reaction; foam-cell clustering; focal mural infiltration; lobular septal cell proliferation; peribronchial alveolar wall epithelization; granulomatosis and neoplasia
	47 rats[f] (250-280 g)	10,000 μg/m³ (430 μg Be) in H_2O	6 hr/day, 5 days/wk (95 days)	1.5	23 deaths; 28% body weight gain; leukocytosis

(continued)

TABLE 13 (Continued)

PHYSIOLOGICAL CHANGES AND MORTALITY RESULTING FROM INHALATION OF BERYLLIUM FLUORIDE, BERYLLIUM OXIDE, AND BERYLLIUM SULFATE[5]

Substance	Animal	Dose	Exposure Duration	Particle Size μ	Effects
Beryllium sulfate	2 swine	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	No deaths; 28% body weight loss
	4 chickens, young adults	47,000 μg/m³ (2,000 μg Be) in H_2O	6 hr/day (51 days)	0.96	No deaths; 11% body weight loss

[a]Data also apply to other young adult animals: 2 cats, 10 dogs, 20 mixed English guinea pigs, 2 rhesus monkeys, and 9 New Zealand rabbits.

[b]Special grade of BeO; 68% of rats, exposed to 82,000 μg/m³ for 15 days, died; all other treated animals survived.

[c]Refractory grade GC of BeO; all animals survived.

[d]Fluorescent grade of BeO; 5% of rats, exposed to 87,000 μg/m³ for 10 days, died; all other treated animals survived.

[e]Refractory grade SP of BeO; all animals survived.

[f]Inhalation of HF vapor (8,000 μg/m³) doubles toxicity of $BeSO_4$ poisoning.

TABLE 14

PHYSICAL PROPERTIES OF BERYLLIUM[7]

Density (specific gravity)	1.846 to 1.816 g/cc[a]
Hardness	97-172 Brinell
Tensile properties[b]	
Ultimate strength	35,000-95,000 lb/inch^2
Yield strength, annealed (offset, 0.2 percent)	30,000-45,000 lb/inch^2
Elongation (in 2 inches)	2-20%%
Reduction of area	2-20%
Young's modulus of elasticity	36,000,000-44,000,000 lb/inch^2
Compressive properties	
Yield strength (offset, 0.2 percent)	26,000-86,300 lb/inch^2
Young's modulus of elasticity	42,000,000-45,000,000 lb/inch^2
Shearing strength	31,000-66,000 lb/inch^2
Electrical properties	
Electrical conductivity	40-44% of Cu
Electrical resistivity	3.9-4.3 microhms/cc
Electrode potential	minus 1.69
Thermal properties	
Melting point	1.285°C (2,345°F)
Boiling point	2,970°C (5,378°F)
Linear coefficient of expan ion:	
(20°-200°C)	13.3 x 10^{-6} per °C
(20°-700°C)	17.8 x 10^{-6} per °C
Thermal conductivity (at 20°C)	0.385 cal/cm^2/cm/sec/°C or 42% of Cu
Specific heat (at 20°-100°C)	0.43-0.52 cal/g/°C
Latent heat of fusion	250-277 cal/g
Vapor pressure: (at 1,400°C)	0.001 mm Hg
(at 3,000°C)	760.0 mm H
Heat of oxidation	140.15 cal[c]
Magnetic susceptibility	0.79 G/O
Reflectivity (white light)	50-55%
Velocity of sound	12,600 m/sec

[a]Theoretical density reported by two different sources.

[b]Tensile properties may vary over a wide range, according to the method of fabrication employed.

[c]Temperature is not given. Value is probably meant to be in kilocalories.

APPENDIX

TABLE 15. CONCENTRATION OF BERYLLIUM[a] IN THE AIR[4]
(in μg/m³)

Location	1954-59[b]		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Alabama												
Birmingham	.001										.000	
Huntsville									.003			
Arizona												
Phoenix			.000				.000				.001	
Tucson			.000									
California												
Los Angeles	.000		.001		.000		.000		.000			
Pasadena							.000					
San Francisco							.000		.000		.000	
Santa Barbara							.000					
Colorado												
Denver	.000						.000		.000		.000	
Pueblo									.000			
Connecticut												
Norwich									.000			
Waterbury	.002											
Delaware												
Wilmington			.000									
District of Columbia												
Washington							.000		.000		.000	
Florida												
St. Petersburg							.000					
Tampa	.000											
Georgia												
Atlanta	.000								.000			
Idaho												
Boise			.000				.000				.000	
Illinois												
Chicago	.000				.001				.000		.001	
Cicero							.001					
East St. Louis									.001			
Joliet									.000			
North Chicago									.000			
Springfield							.000					

(continued)

APPENDIX

TABLE 15. CONCENTRATION OF BERYLLIUM[a] IN THE AIR[4] (Continued)
(in μg/m³)

Location	1954-59[b]		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Indiana												
E. Chicago											.005	
Gary			.000									
Indianapolis	.000				.003		.000				.001	
Muncie									.000			
Park County											.000	
Iowa												
Cedar Rapids									.000			
Des Moines	.000		.000				.000				.000	
Kansas												
Wichita	.000										.000	
Louisiana												
Lake Charles							.000					
New Orleans	.000						.000		.000			
Maryland												
Baltimore	.000		.000		.000		.000				.001	
Cumberland									.000			
New York												
Buffalo							.000					
Glen Cove							.000					
Massena					.000							
Mount Vernon					.000							
New Rochelle					.000							
New York					.000		.000		.000		.000	
Rochester					.000							
Troy							.000					
North Carolina												
Asheville			.000									
Charlotte	.000										.000	
Durham												
Winston-Salem							.000					
North Dakota												
Bismarck	.000											

(continued)

TABLE 15. CONCENTRATION OF BERYLLIUM[a] IN THE AIR[4] (Continued)
(in μg/m³)

Location	1954-59[b]		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Ohio												
Akron			.001									
Canton					.000							
Cincinnati			.002		.000		.000		.001		.001	
Cleveland	.000						.001		.002		.000	
Columbus					.002		.000					
Dayton			.002									
Youngstown			.000									
Oklahoma												
Oklahoma City					.000		.000					
Tulsa					.000							
Michigan												
Detroit	.000						.002		.000		.001	
Grand Rapids									.000			
Minnesota												
Minneapolis	.000										.000	
St. Paul							.000					
Missouri												
St. Louis	.000						.000		.000		.000	
Montana												
Helena											.000	
Nebraska												
Omaha	.000				.000		.000					
Nevada												
Las Vegas					.000		.000		.000		.000	
Reno									.000			
New Hampshire												
Manchester	.000											
New Jersey												
Bayonne								.000				
Camden							.000					
Glassboro											.000	
Newark					.001		.000				.000	
Paterson									.000			

(continued)

TABLE 15. CONCENTRATION OF BERYLLIUM[a] IN THE AIR[4] (Continued)
(in μg/m³)

Location	1954-59[b]		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
New Mexico												
Albuquerque			.000									
Oregon												
Eugene					.000							
Medford					.000							
Portland	.000										.000	
Pennsylvania												
Allentown					.003							
Altoona									.000			
Bethlehem					.000							
Lancaster							.000					
Philadelphia	.000		.001		.000		.000		.001		.000	
Pittsburgh			.000				.000		.001		.000	
Scranton					.002							
Rhode Island												
East Providence									.000			
Tennessee												
Chattanooga					.000						.000	
Nashville	.000										.000	
Texas												
El Paso							.000				.000	
Houston											.000	
Odessa									.000			
Tyler									.000			
Utah												
Salt Lake City	.000										.000	
Washington												
Seattle	.000						.000		.000			
Spokane							.000					
Tacoma	.000											
West Virginia												
Charleston	.004											
Huntington			.000									

(continued)

TABLE 15. CONCENTRATION OF BERYLLIUM[a] IN THE AIR[4] (Continued)
(in ug/m³)

	1954-59[b]		1960		1961		1962		1963		1964	
	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg	Max	Avg
Wisconsin												
Milwaukee	.000										.000	
Racine			.000									
Wyoming												
Cheyenne	.000											

[a]Values of 0.000 represent undetectable amounts.

[b]The data in this column may include only 1 year or the average of all measurements made during these years.